



47.1 Introduction

Maternal cardiovascular disease complicates approximately 0.2–4% of all pregnancies in western industrialized countries, and it is the major cause of maternal death during pregnancy. Women are increasingly seeking pregnancy at a later age characterized by cardiovascular risk factors (diabetes, obesity, hypertension), resulting in more frequent cardiac events. Furthermore, surgery of congenital heart disease has improved, so as an increased number of women with congenital problems contemplate pregnancy.

47.2 Haemodynamic Changes/ Physiologic Alterations

Pregnancy is a physiological state that induces adaptation of the cardiovascular system to meet the increased metabolic demands of the mother and foetus and to ensure adequate uteroplacental circulation for foetal growth and development. These changes, which occur during labour and delivery and in the postpartum period, can mimic cardiac disease so that an understanding of both

haemodynamic alterations and anatomic changes is the key to interpret the echocardiograms in pregnant patients. Further, the normal haemodynamic changes can precipitate cardiac symptoms in previously stable women or may exacerbate symptoms in those with mild baseline symptoms.

Cardiac output increases by about 30–50% by the end of the first trimester, primarily due to an increase in stroke volume and in circulating blood volume. Between the second and third trimesters, cardiac output shows a peak, owing to a 15–20% increase in heart rate and a fall in after-load via decreased total vascular resistance. Arterial blood pressure decreases early in pregnancy, characterized by decreases in DBP exceeding those in SBP. The mean arterial pressure gradually falls, with the largest decrease occurring at 16–20 weeks. BP then begins to rise during the mid-third trimester to levels approaching pre-pregnancy BP values.

During labour and delivery, pain and uterine contractions result in additional increases in cardiac output (20% with each contraction), heart rate, blood pressure and systemic vascular resistance. Because of contribution of pain and anxiety to the increase in heart rate and blood pressures, pain control may help mitigate the haemodynamic changes. Immediately following delivery, relief of caval compression and auto-transfusion from the emptied and contracted uterus results in a further increase in preload with

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venous return of blood to the maternal circulation (approximately 500 mL). This effect can cause cardiac output to increase by 60–80%. Delivery of the placenta increases afterload by removing this low-resistance vascular bed. Most haemodynamic changes of pregnancy resolve by 2 weeks postpartum.

47.3 Heart Remodelling

During normal pregnancy, the progressive and marked increase in left ventricular stroke volume (as a high-output state) can result in an increased velocity across valves, which may falsely increase systolic or diastolic gradients. The normal flow murmur of pregnancy is typically soft (grade 1 or 2), located at the pulmonic region, associated with a normal first and second heart sound, and is not accompanied by a diastolic murmur or signs of heart failure. As the heart enlarges, the mitral, tricuspid and pulmonary annuli dilate and small functional atrioventricular regurgitations may appear through these valves. As left ventricular volume increases and the position of the mitral valve leaflets changes with respect to one another, pre-existing mitral regurgitation due to mitral valve prolapse may actually improve during pregnancy. During gestation, a small pericardial effusion haemodynamically non-significant is not necessarily indicative of pathology.

47.4 Echocardiography

Echocardiography is, by far, an important tool during pregnancy and the preferred diagnostic exam to evaluate pregnant women with cardiac disease due to its availability, accuracy and safety with no risk of radiation to the mother and the foetus. A transthoracic echocardiogram provides both qualitative and quantitative information; in addition it can help to stratify maternal risk during pregnancy and consequently to choose the correct management. Depending on the underlying cardiac abnormality, serial echo examinations rather than a single study may be indicated.

The following conditions are indications to perform an echocardiogram in a pregnant woman:

1. Cardiac complaints including shortness of breath and palpitations out of proportion to what is expected in a normal pregnancy to exclude a diagnosis of a new cardiac disease
2. Pre-existing hypertension to evaluate systolic and diastolic cardiac function
3. Known heart disease (congenital heart disease, cardiomyopathy, ischemic heart disease, valvular heart disease, arrhythmia) to exclude residual defects that need intervention and to evaluate maternal risk
4. Known aortic root dilatation to stratify risk of rupture and decide management
5. Stroke of unknown aetiology
6. A prior history of chemotherapy or radiation in a woman to evaluate cardiac function

MRI (without gadolinium) should be considered if echocardiography is insufficient for diagnosis, and it can be safely performed after the first trimester.

47.4.1 Echocardiographic Findings

The echocardiographic changes during pregnancy are based on the morphological and physiological adaptation of the human heart to transient preload and afterload changes. The main features are detailed in Table 47.1.

Healthy pregnancy is associated with prolonged cardiac volume overload secondary to increased blood volume that results in a reversible “physiologic” eccentric left ventricular (LV) hypertrophy with the aim of preserving adequate oxygen supply. Starting from as early as 12 weeks of gestation, LV wall thickness increases by about 15–25% to minimize wall stress and maintain myocardial oxygenation; LV wall mass increases by about 50% above pre-pregnancy values mainly during the third trimester (increase in both LV mass and left ventricle mass index). The LV mass increases result from the increased LV end-diastolic and end-systolic diameters (about 12% and 20%, respectively), increased LV posterior wall diastole and systole (about 22%

Table 47.1 Echocardiographic findings during pregnancy

Echocardiographic variables	Changes during pregnancy
Left ventricular dimension and volume	Increases
Left ventricular wall thickness and left ventricular mass	Increases
Left ventricular ejection fraction	Unchanged
Left ventricular fractional shortening	Unchanged
Left ventricular radial and longitudinal strain rate	Increases
Aortic root diameter	Mildly increases
Right ventricular dimension and volume	Increases
Right ventricular ejection fraction	Unchanged
Left atrial size and volume	Increases
Stroke volume (as measured using pulsed wave Doppler)	Increases
Mitral E wave velocity	Increases and then decreases
Mitral A wave velocity	Increases
Peak pulmonary artery systolic pressure estimated using tricuspid regurgitation jet	Unchanged

and 13%, respectively) and intraventricular septal thickness during diastole and systole (about 15% and 19%, respectively). Similar to the LV, the right ventricle (RV) increases in size throughout pregnancy because of increased preload. Sphericity index decreases during pregnancy, indicating a more spherical shape of the left chamber towards the third trimester. This physiologic cardiac remodelling resolves in the first weeks postpartum.

47.4.1.1 Left Atrium

Left atrium acts as a reservoir, a conduit and a booster pump during the cardiac cycle. The left atrium dimensions and function are enhanced gradually in pregnancy, as an adaptive response to increased preload. These changes cause a gradually improved atrial contribution to ventricular filling, so as to maintain adequate LV stroke volume and cardiac output. These findings result in a slightly lower tissue Doppler *E* velocity and ascended tissue Doppler *A* wave. The speckle-tracking echocardiography as well shows an improved left atrium reservoir and booster pump function; concerning the conduit function the speckle-tracking echocardiography shows it is decreased. However, all these changes are reversible.

47.4.1.2 Left Ventricular Function

The data regarding systolic LV function during normal pregnancy are conflicting. The debate is still open whether the LV function deteriorates as a consequence of the physiological changes during pregnancy (comparable to exercise-induced cardiac remodelling) or has no changes or is enhanced in pregnancy. Regarding diastolic function, *E* wave and *A* wave are increased, as a result of the over preload and increased blood volume. The *E/A* ratio and *E'/A'* ratio decrease, while the *E/E'* remains into normal range.

47.4.1.3 Right Ventricular Function

There are too few echocardiographic studies that evaluate RV function during normal pregnancy.

47.5 Hypertensive Disorders

Paradigmatic examples of the utility of echocardiography in pregnancy are the hypertensive disorders. Echocardiography has the potential to categorize patients with gestational hypertension or preeclampsia into high- and low-risk groups. Women with increased LV mass are more likely to have complications. Reduced *e'* and therefore elevated *E/e'* may be an early predictor of preeclampsia. Echocardiography can also help to identify the small numbers of women with LV systolic dysfunction who are more likely to deteriorate during pregnancy or postpartum. In addition the use of 3D speckle tracking confirms the known LV remodelling and changes in LV function and allows additional detection of earlier abnormal radial and longitudinal strain values in early preeclampsia compared to late preeclampsia.

47.6 Valvular Heart Diseases

The main features are detailed in Table 47.2.

47.7 Congenital Heart Diseases

The main features are detailed in Table 47.3.

Table 47.2 Management of pregnant women with valvular diseases

Valvular disease	Pathophysiology	Considerations	Diagnostic assessment	Management	Labour and delivery
Mitral stenosis (MS)	Elevated transmitral gradient due to the physiological rise in blood volume, cardiac output and heart rate Increase in left atrial pressure and left atrial dilatation can cause arrhythmias Cardiac complications: dyspnoea, decreased exercise capacity, orthopnoea, paroxysmal nocturnal dyspnoea, pulmonary oedema or less frequently arrhythmias	Gradient and PAP have a prognostic value (do not directly reflect the severity of MS during pregnancy) Guidelines advise against pregnancy for a woman with valve area <1.1 cm ² or who has significant pulmonary hypertension Haemodynamic deterioration more often happens towards the end of the second or beginning of the third trimester, when maternal blood volume and cardiac output peak	Echocardiography to confirm the diagnosis and to determine the severity of the stenosis and assessments of pulmonary function, right ventricular function, mitral regurgitation and the configuration of the subvalvular apparatus Monthly/bimonthly clinical and echocardiographic follow-up, depending on haemodynamic tolerance Trimester evaluation and prior to delivery in mild MS Invasive diagnostic testing is rarely indicated	NYHA class II to IV symptoms or severe pulmonary hypertension (defined as pulmonary artery pressure >75% of systemic pressure) should be referred for prophylactic percutaneous mitral balloon valvotomy (PMBV) or open commissurotomy before conception Medical therapy directed at the treatment of volume overload (diuretics) and to attenuate the increases in heart rate and prolong the diastolic filling period (β-blockers)	Vaginal delivery (shortening of the second stage of labour and assisted delivery) in mild MS and in moderate or severe MS in NYHA I/II without pulmonary hypertension Epidural analgesia to reduce fluctuations in HR and CO Caesarean section for obstetric considerations and in moderate or severe MS in NYHA III/IV or with pulmonary hypertension despite medical therapy Invasive haemodynamic monitoring for women with moderate to severe MS or symptoms of heart failure During the first two stages, successive uterine contractions lead to an increase in atrial pressures The most vulnerable time is immediately after delivery: an abrupt increase in venous return, and therefore left atrial pressure, may lead to acute pulmonary oedema (haemodynamic monitoring for 12–24 h after the delivery)
Aortic stenosis (AS)	Increased gradient across the aortic valve due to an increase in stroke volume and in CO and a fall in peripheral resistance The clinical consequences of the increased aortic gradient depend on the degree of pre-existing LV hypertrophy and LV systolic function	Mild-to-moderate AS with preserved LV function usually is well tolerated during pregnancy Symptoms (dyspnoea, angina pectoris, syncope) usually become apparent late in the second trimester or early in the third trimester	Echocardiography can confirm diagnosis, detect LV hypertrophy and estimate ejection fraction and LV dimensions Monthly/bimonthly cardiac evaluations in severe AS Women with EF <55% are at high risk for developing heart failure during pregnancy In case of a congenital maternal AS, foetal echocardiography is indicated	Exercise testing is recommended in asymptomatic patient before pregnancy (to evaluate exercise tolerance, BP response, arrhythmias) Medical therapy (preload reduction) Aortic valve replacement and palliative aortic balloon valvuloplasty (with associated maternal and foetal risk)	Vaginal delivery with assisted second stage of labour in non-severe AS, avoiding a decrease in peripheral vascular resistance in case of regional analgesia Caesarean delivery with general anaesthesia is recommended in severe symptomatic AS (intrapartum increases in CO related to contractions may significantly increase the risk of cardiac events) Haemodynamic monitoring is strongly recommended for labour and delivery in moderate and severe AS

<p>Mitral and aortic regurgitation</p>	<p>Reduced regurgitant volume due to the physiological fall of systemic vascular resistance, both from widespread vasodilatation and by virtue of the low-resistance placental circulation The increase in blood volume is likely only to cause a modest increase in pressure of an already dilated left atrium</p>	<p>Generally well tolerated Severe regurgitation with LV dysfunction is poorly tolerated (high risk of heart failure)</p>	<p>Doppler echocardiography is useful in diagnosis, evaluation of the structure of the valve and subvalvular apparatus (papillary muscle, chordae tendineae), assessment of left ventricular size and function, left atrial size and left atrial appendage thrombosis Every trimester follow-up in mild/moderate regurgitation, more often in severe regurgitation</p>	<p>Medical therapy Surgical repair Women with pulmonary arterial pressure greater than 50 mm Hg are at increased risk for complications</p>	<p>Vaginal delivery Epidural analgesia and shortened second stage in symptomatic patients</p>
<p>Pulmonary stenosis (PS)</p>	<p>Generally well tolerated Severe stenosis may result in RV failure and arrhythmias Increased incidence of maternal obstetrics complications, particularly hypertension-related disorders including (pre-)eclampsia</p>	<p>In mild and moderate PS (low-risk lesions): follow-up once every trimester In severe PS: monthly/bimonthly cardiac evaluation including echocardiography (RV function)</p>	<p>Balloon valvuloplasty should be performed in severe stenosis (peak Doppler gradient >64 mmHg)</p>	<p>Vaginal delivery in non-severe PS or severe PS in NYHA class I/II Caesarean section in severe PS and in NYHA class III/IV</p>	
<p>Pulmonary regurgitation</p>	<p>Independent predictor of maternal complication, especially in case of concomitant impaired ventricular function</p>	<p>Pre-pregnancy valve replacement in symptomatic women or in RV abnormal function due to severe pulmonary regurgitation</p>			<p>Vaginal delivery</p>
<p>Tricuspid regurgitation</p>					

Table 47.3 Management of pregnant women with congenital heart diseases

Type of lesion	WHO* class I	WHO *class II	WHO *class III	WHO* class IV
	Mild pulmonary stenosis Small patent ductus arteriosus (PDA) Repaired atrial septal defect (ASD) Repaired ventricular septal defect (VSD) Repaired PDA Repaired total anomalous venous drainage	Unrepaired ASD Unrepaired VSD Repaired tetralogy of Fallot (TOF) Repaired aortic coarctation (COAO)	Systemic right ventricle (RV): e.g. atrial switch with Mustard or Senning for TGA Fontan operation for univentricular heart Complex CHD repaired Cyanotic CHD	Pulmonary hypertension Native severe aortic coarctation
Physiopathology	As normal pregnancy	Left to right shunt (ASD, VSD) Risk of pulmonary hypertension (ASD, VSD) Obstruction to pulmonary flow with high pressure in RV (TOF repaired) RV function (ASD, TOF) Arrhythmias (ASD, TOF) Hypertension (COAO) Every trimester, to rule out residual lesions and evaluate heart function	Right to left shunt RV function Arrhythmias Coronary problems (in particular TGA) Embolism	
Echo	As normal pregnancy		Every 2 weeks or monthly, to rule out residual lesions and evaluate heart function	
Pregnancy	Not avoided	Low to moderate risk	High risk	Counsel to avoid pregnancy
Delivery	Vaginal delivery	Vaginal delivery		
Management	Not necessarily in a specialized centre with a specialized team	Counselling and first visit in a specialized centre with a congenital grown up (GUCH) unit. If not high risk pregnancy or residual lesion, delivery can happen also in a centre without a specialized team	Only in specialized centre with a GUCH unit	
<i>General considerations:</i>				
Vaginal delivery is preferable and safer Labour may be induced with good pain control (incremental epidural analgesia) Labour can be conducted with the mother in left lateral position to avoid inferior caval compression and maintain venous return Maternal ECG monitor should be performed to detect any arrhythmia during labour and puerperium Continuous pulse oximetry is useful Intra-arterial monitors can be an option in some circumstances (e.g. cyanotic heart disease) Caesarean section (preferably planned) in case of: Obstetric consideration Patient on Coumadin (to avoid risk of foetal intracranial haemorrhage) Severe obstructive lesions (to minimize the haemodynamic perturbations) Aortopathy with unstable aorta (to minimize the haemodynamic perturbations) Severe pulmonary hypertension				
The duration of postpartum monitoring must be individualized. For WHO class III or IV should be continued at least 24 h after delivery in an ICU				

Suggested Reading

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